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RESPIRATION ON THE LEFT AND
RIGHT VENTRICLES

A DISSERTATION SUBMITTED TO THE
FACULTY OF THE DIVISION OF THE
BIOLOGICAL SCIENCES IN CANDIDACY FOR
THE DEGREE OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF PHYSIOLOGY

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The systemic arterial blood pressure changes (fall in inspiration, rise in expiration) have commonly been thought to be due in part to fluctuations in the filling and consequently the output of the heart induced by respiratory fluctuations in intrathoracic pressure. The decreased intrathoracic pressure associated with inspiration is generally considered to have an "aspiratory" effect upon the veins within the thorax, and hence to augment the inflow of blood to the right heart (Burton-Opitz, 1902; Hooker, 1921; Wiggers, 1921; Visscher, Rupp and Scott, 1924; Heinbecker, 1927). Closed chest measurements of cardiac output by means of a glass oncometer similar to that first described by Wiggers and Katz (1922) have given rather contradictory results. Eyster and Hicks (1933) found that inspiration was associated with greater diastolic size, but with a decreased stroke volume, which seemed inconsistent with the Starling principle. Cahoon, Michael and Johnson (1941) found that there were both decreased diastolic size and decreased stroke volume during inspiration. These findings were consistent with the Starling principle, and seemed to account, in part, for the fall in arterial pressure during inspiration.

Boyd and Patras (1941), recognizing the fact that previous measurements of cardiac output had been made with the ventricles operating against an unchanging atmospheric pressure, designed a recording system wherein the ventricles operated against fluctuating negative pressures approximating those of the closed chest of a dog breathing normally. Their experiments showed that when the heart operated against approximately intrathoracic pressures instead of atmospheric pressures there was a definite increase of both diastolic volume and stroke volume during inspiration. These results were consistent with both the Starling principle and the concept of aspiration of blood into the thorax and heart in inspiration, but left unanswered this question: if the heart pumps more blood during inspiration, why should the arterial blood pressure fall during that phase of respiration?

EXPERIMENTS EMPLOYING THE DIFFERENTIAL MANOMETER. In an attempt to check the findings of Boyd and Patras, a recording system was designed which allowed free interchange of pressure variations between the interior of the chest and the ventricles in the oncometer, and which in addition eliminated the possibility of any respiratory displacement of the recording membrane. The method of recording was adapted to the stationary optical manometer devised by Hamilton, Brewer and Brotman (1934). The manometer was of the differential

type, equipped with a very sensitive rubber membrane (see fig. 1, right). One chamber of the manometer was connected to the oncometer, the other to the intrapleural space. A communication was established between the two tubes leading to the chest and the oncometer respectively. This communicating tube could be constricted or enlarged at will, thus affording a means of regulating the excursion of the recording membrane. (The oncometer system was also con-

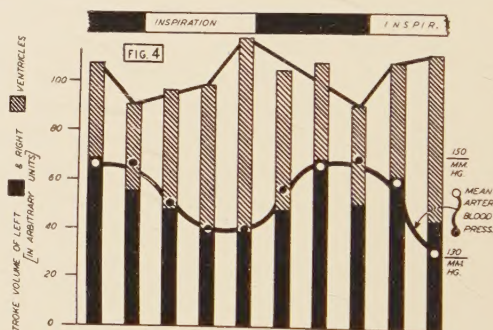
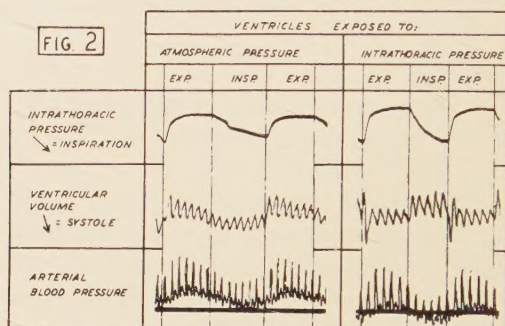
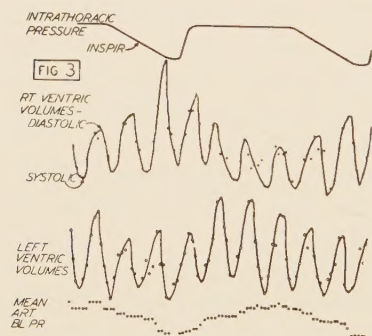
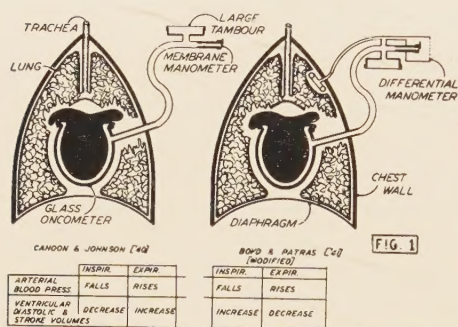


Fig. 1. Diagrammatic representation of cardiometer recording system. At left is system used by Cahoon, Michael and Johnson; at right is system described in this paper.

Fig. 2. Simultaneous curves of intrathoracic pressure, ventricular volume, and carotid pressure. At left with the ventricles under atmospheric pressure, at right with ventricles under intrathoracic pressure.

Fig. 3. Graph of intrathoracic pressure, right and left ventricular "volumes," and carotid pressure. Ordinates for ventricular volumes are measured areas in square inches, abscissas are $\frac{1}{32}$ second intervals (movies at 32/sec.).

Fig. 4. Block diagram of same record as figure 3, showing right, left, and total ventricular stroke volumes in ten successive heart beats occurring during nearly two respiratory cycles. Mean carotid blood pressure is superimposed.

nected to the usual large closed air space which allowed a minimum of interference with cardiac filling and ejection.) It can be seen that this system allowed free interchange of pressure variations between the chest and the oncometer, and that at the same time the influence of intrathoracic pressure was equal on both sides of the recording membrane. By sufficiently constricting the intercommunicating tube, a condition could be obtained in which the relatively slow

chest pressure changes were distributed throughout the whole system, while the rapid movements of the heart could still cause excursions of the manometer membrane. Controls run during each experiment showed that there was no displacement of the membrane due to respiratory pressure changes. Recording could be done under the conditions used by Cahoon, Michael and Johnson (1941) (fig. 1, left) by closing the intercommunicating and intrapleural tubes. Direct comparisons of the two methods could be made from these records.

Medium-sized dogs anesthetized with sodium barbital were used. Artificial respiration was supplied by means of interrupted blasts, the 4th and 5th ribs on the right were resected, the pericardium removed, and an oncometer was placed over the two ventricles. Care was taken to assure that there was neither leakage nor constriction at the A-V groove. Before closing the chest, two metal trocars were inserted between the 2nd and 3rd ribs, one on either side of the midline. One of these trocars was attached directly to an optical manometer for recording intrathoracic pressure changes, and provided with a side tube through which air could be exhausted from the chest. The other trocar and the oncometer were attached to the differential manometer as described in the preceding paragraph. The chest was closed with large hemostats, the air was aspirated from the chest, and normal respiration allowed to proceed. Carotid pressures were recorded simultaneously.

Figure 2 shows records taken under the two different recording conditions, within approximately thirty seconds of each other. These results (right half of figure) confirm those of Boyd and Patras (1941) even to the point that the largest systolic excursion occurred when expiration and systole began simultaneously. No accurate calibration of the volume changes of the heart was obtainable with this recording system although a very crude calibration could be made at the close of the experiment by rapidly injecting into or removing from the oncometer tubing, known volumes of air.

Figure 2 demonstrates that carotid blood pressure dropped during inspiration whether the ventricles were exposed to atmospheric or intrathoracic pressure. In these and all previous records made with the ventricles under atmospheric pressure (fig. 2, left) the rise and fall in carotid pressure corresponded to the rise and fall of ventricular diastolic volume and stroke. But if the results obtained with the ventricles under intrathoracic pressure (fig. 2, right) are valid, it becomes necessary to explain why in inspiration the carotid pressure falls when the ventricles are ejecting more blood. It was thought that perhaps at the time of inspiration there might be an *increase* in the diastolic and stroke volumes of the *right* ventricle which would be so large as to mask a possible *decrease* in the *left* heart volume and stroke. Since no satisfactory method has been evolved to measure directly the volume changes of the ventricles independently of each other, the following experiment was devised:

Differential ventricular volumes. Large dogs, (18-25 kgm.) were used, anesthetized with sodium barbital. A ventral "plate" of ribs and muscle was removed from directly over the heart. A crucial incision was made in the pericardium and the edges were sutured back to the chest wall to form a cradle for the heart.

Small discs of white cardboard with black centers were affixed to the ventricles with collodion to outline each ventricle as completely as possible. After inserting a trocar through the chest wall for recording of respiration, a curved window of transparent plastic was placed over the chest opening, and the skin pulled up around it to form an air tight seal. The chest was evacuated and normal respiration allowed to proceed. Indicators, for recording carotid and intrathoracic pressures, were arranged to move above suitable scales within the optical field of a camera aimed at the heart. High speed motion pictures were taken under different experimental conditions.

Individual photographic enlargements were then made of a number of successive frames from each experiment. Each group of approximately 140 frames included at least two respiratory cycles. Lines were drawn between the markers on each print to outline each ventricle separately, using the intraventricular septum as common to both ventricles. The enclosed areas in each frame were measured with a planimeter and simultaneous blood and intrathoracic pressures observed. Only groups of frames were used where careful counts showed the heart rate to be constant. Figure 5 shows four frames representative of the comparative ventricular areas at the peak of systole and diastole during the height of inspiration and expiration respectively¹.

It must be remembered that it is *not* assumed that the measured areas are accurate reflections of the volumes of the ventricles, but to avoid coining new and useless names the terms "volume", "stroke" and "output" will be applied without modification. All numerical results are in arbitrary units, but they are strictly comparable in each experiment, since projection distances were kept constant throughout. The measured areas of the right and left ventricle were never more than approximately equal, but it was assumed that the output of the left ventricle would be equal to that of the right over a period of several respirations, and numerical results were in some cases corrected and graphing was done accordingly. However, the graphs showed the same qualitative results whether crude or corrected data were used.

Figure 3 gives the results of one experiment in which are plotted the measurements, from the successive movie frames, of left and right ventricular "volumes," intrathoracic pressure, and mean arterial blood pressure. It can be seen from this and the numerical data given in figure 5 that during inspiration the diastolic and stroke volumes in the right ventricle increase, while the corresponding measurements of the left ventricle decrease. The reverse is true during expiration. This corresponds with the findings of Cahoon, Michael and Johnson (1941) on the auricles: in inspiration the diastolic volume of the right auricle increases, while that of the left auricle decreases. Figure 4 shows that when measurements are made with the ventricles under intrathoracic pressure, the total stroke output increases during inspiration. This is due to the great increase in the stroke of the right ventricle, which is sufficiently great to mask the simultaneous diminution in left ventricular stroke volume. The mean arterial

¹ These prints were made directly from positive film, so the blacks and whites are naturally reversed.

blood pressures, when graphed on an appropriate scale, coincide at the majority of points with the changes in left ventricular stroke volumes (as would be expected) and are independent of the activity of the right ventricle. The effects of respiratory changes on ventricular volume and stroke were augmented by deep respiration and tracheal occlusion, diminished during quiet, shallow breathing, and eliminated during apnea induced by central superior laryngeal stimulation.

Differential blood pressures. To determine whether intrathoracic pressure changes are directly transmitted to the systemic arteries, contributing to the

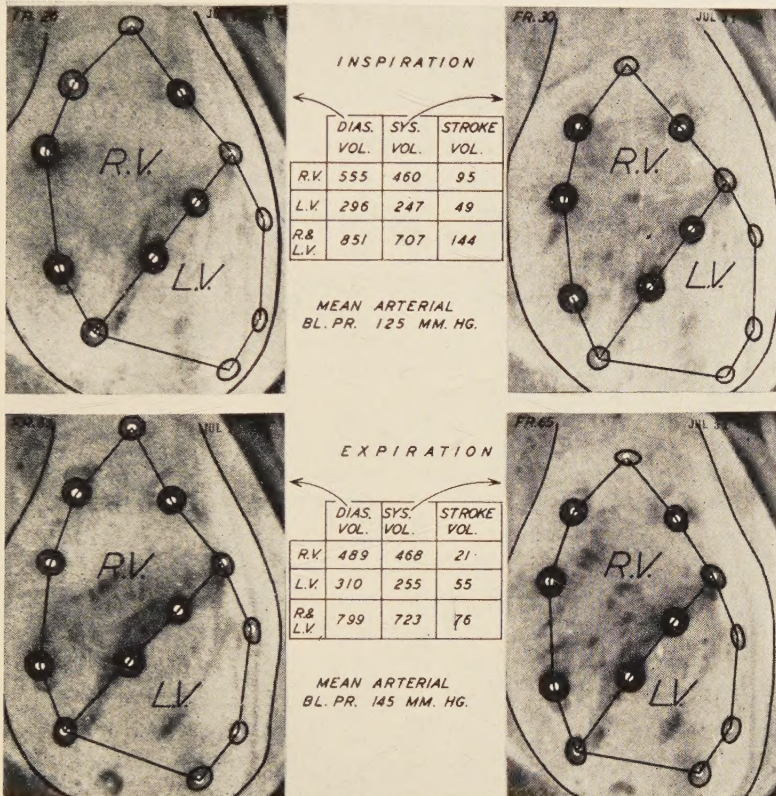


Fig. 5. Photographs of four selected movie frames, showing method of measuring areas of ventricles.

respiratory changes in arterial pressure, the following experiments were devised: Four dogs were trained daily to lie quiescent while femoral blood pressures were taken by direct arterial puncture. Simultaneous pneumograph and blood pressure recordings were taken on the Hamilton manometer. The animals showed definite sinus arrhythmia and a distinct drop in blood pressure which coincided with inspiration. The arrhythmias and respiratory variations were present even after the dogs had become accustomed to the procedure, at which time breathing was normal. Other records from animals which had been trained for

5. Systemic arterial blood pressure measurements on trained, unanesthetized dogs showed but slight reduction of respiratory influences on blood pressure when intrathoracic pressure influences were eliminated by the use of a differential manometer. These reductions are equivalent to simultaneous intrathoracic pressures, but are not sufficient to eliminate respiratory fluctuations in blood pressure.

6. The main factor responsible for the arterial blood pressure fluctuations of respiration is the changing output of the left ventricle. A sinus arrhythmia and direct influences of intrathoracic pressures may modify somewhat these blood pressure changes.

7. Each ventricle responds independently in accordance with the Starling principle, regardless of the diastolic size of the other ventricle.

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